APPENDIX (Pending Claims)

1. (Previously presented) A method of enhancing an endogenous immune response-mediated specific elimination of a population of cancer cells in a host animal harboring said population wherein the members of said cell population have an accessible binding site for a folate receptor-binding ligand, said method comprising the step of

administering to said host a composition comprising an immunogen conjugated to a folate receptor-binding ligand selected from the group consisting of folate and analogs and derivatives thereof wherein the immunogen is recognized by an endogenous or an exogenous antibody in the host or is recognized directly by an immune cell in the host; and

administering to said host a compound capable of stimulating an endogenous immune response wherein the compound does not bind to the conjugate.

2. (Canceled)

3. (Canceled)

4. (Withdrawn) The method of claim 1 wherein the population of pathogenic cells is an exogenous pathogen or an endogenous cell population harboring exogenous pathogens.

5. (Withdrawn) The method of claim 4 wherein the exogenous pathogen is selected from the group consisting of bacteria, fungi, viruses, mycoplasma, and parasites.

6. (Canceled)

7. (Canceled)

8. (Previously presented) The method of claim 1 wherein the folate receptor-binding ligand is chemically complexed to the immunogen through bonding selected from the group consisting of covalent, ionic, and hydrogen bonding.

9. (Previously presented) The method of claim 8 wherein the folate receptor-binding ligand is a folic acid analog having a glutamyl moiety covalently linked to the immunogen only via the glutamyl γ-carboxyl moiety of the ligand.

10. (Previously presented) The method of claim 8 wherein the folate receptor-binding ligand is a folic acid analog having a glutamyl moiety covalently linked to the immunogen only via the glutamyl α-carboxyl moiety of the ligand.

11. (Canceled)

12. (Canceled)

13. (Previously presented) The method of claim 1 wherein the ligand is an organic molecule capable of binding to a receptor and wherein said receptor is preferentially expressed, uniquely expressed or overexpressed on the surface of said population of cancer cells.

14. (Withdrawn) The method of claim 12 wherein the small organic molecule is an antimicrobial drug.

15. (Withdrawn) The method of claim 1 wherein the ligand is a ß-lactam antibiotic.

16. (Cancelled)

17. (Canceled)

18. (Original) The method of claim 1 wherein the immunogen is an organic molecule having a molecular weight less than 20,000 daltons.

19. (Previously presented) The method of claim 18 wherein the organic molecule is fluorescein or dinitrophenyl.

20. (Original) The method of claim 1 wherein the immunogen is an α-galactosyl group.

21. (Original) The method of claim 1 wherein the antibody is exogenous to said host and is co-administered with said conjugate composition.

22. (Previously presented) The method of claim 1 wherein the compound capable of stimulating an endogenous immune response comprises a cytokine.

23. (Previously presented) The method of claim 22 wherein the cytokine comprises IL-2, IL-12, IL-15, or combinations thereof.

24. (Previously presented) The method of claim 22 wherein the cytokine comprises IL-2, IL-12, IL-15, or combinations thereof, in combination with IFN-α or IFN-γ.

25. (Previously presented) The method of claim 22 wherein the cytokine comprises IL-2, IL-12, IL-15, or combinations thereof, in combination with IFN-α or IFN-γ, or a combination thereof, and GM-CSF.

26. (Previously presented) The method of claim 1 wherein the compound capable of stimulating an endogenous immune response comprises at least one NK cell or T cell stimulant.

27. (Previously presented) The method of claim 1 wherein the conjugate composition is administered in multiple injections.

28. (Original) The method of claim 1 wherein the host animal had been previously exposed naturally to the immunogen so that the host animal has a preexisting immunity to said immunogen evidenced by the presence of endogenous antibodies to the immunogen.

29. (Original) The method of claim 1 wherein the host animal had been previously exposed to the immunogen by a non-natural process resulting in priming of the host animal's immune response to said immunogen.

30. (Previously presented) The method of claim 29 wherein the non-natural process resulting in priming of the animal's immune response is vaccination.

31. (Previously presented) The method of claim 29 wherein the non-natural process resulting in priming of the immune response is active immunization.

32. (Original) The method of claim 1 wherein the endogenous immune response comprises a humoral immune response.

33. (Previously presented) The method of claim 32 wherein the humoral response is an acquired immune response.

34. (Previously presented) The method of claim 32 wherein the humoral response is an innate immune response.

35. (Previously presented) The method of claim 33 wherein the acquired response is induced by administering into the host animal a vaccine composition.

36. (Original) The method of claim 1 wherein the endogenous immune response comprises a cell-mediated immune response.

37. (Original) The method of claim 1 wherein the endogenous immune response comprises a humoral and a cell-mediated immune response.

38. (Previously presented) A method of enhancing an endogenous immune response-mediated specific elimination of a population of cancer cells in a host animal harboring said population wherein said population expresses a binding site for a folate receptor-binding ligand, said method comprising the steps of

administering to the host a composition comprising a conjugate of said ligand and an immunogen;

administering to the host antibodies directed against the immunogen; and

administering to said host a stimulant of an endogenous immune response that does not bind to the ligand-immunogen conjugate.

39. (Canceled)

40. (Canceled)

41. (Previously presented) A method of enhancing an endogenous immune response-mediated specific elimination of a population of cancer cells in a host

animal harboring said population wherein said population preferentially expresses, uniquely expresses, or overexpresses a folic acid receptor, said method comprising the steps of

administering to said host a composition comprising a covalently linked conjugate of a ligand and an immunogen wherein the immunogen is recognized by an endogenous or exogenous antibody in the host or is recognized directly by an immune cell in the host;

wherein the ligand comprises folic acid or a folic acid analog having a glutamyl group wherein the covalent linkage is only through the γ- carboxy group of the glutamyl group; and

administering to said host a compound capable of stimulating an endogenous immune response wherein the compound does not bind to the ligand-immunogen conjugate.

42. (Previously presented) A method of enhancing an endogenous immune response-mediated specific elimination of a population of cancer cells in a host animal harboring said population wherein said population preferentially expresses, uniquely expresses, or overexpresses a folic acid receptor, said method comprising the step of

administering to said host a composition comprising a covalently linked conjugate of a ligand and an immunogen wherein the immunogen is recognized by an endogenous or exogenous antibody in the host or is recognized directly by an immune cell in the host;

wherein the ligand comprises folic acid or a folic acid analog having a glutamyl group wherein the covalent linkage is only through the α- carboxy group of the glutamyl group; and

administering to said host a compound capable of stimulating an endogenous immune response wherein the compound does not bind to the ligand-immunogen conjugate.

43. (Previously presented) A pharmaceutical composition comprising therapeutically effective amounts of an immunogen conjugated to a folate receptor-binding

ligand selected from the group consisting of folate and analogs thereof, a compound capable of stimulating an endogenous immune response wherein the compound does not bind to the ligand-immunogen conjugate, and a pharmaceutically acceptable carrier therefor.

44. (Previously presented) The pharmaceutical composition of claim 43 in a parenteral prolonged release dosage form.

45. (Previously presented) The pharmaceutical composition of claim 43 wherein the compound capable of stimulating an endogenous immune response is a cytokine.

46. (Previously presented) The pharmaceutical composition of claim 45 wherein the cytokine comprises a compound selected from the group consisting of IL-2, IL-12, IL-15, IFN-α, IFN-γ, and GM-CSF, or combinations thereof.

47. (Withdrawn) A method of enhancing an endogenous immune response-mediated specific elimination of a population of pathogenic cells in a host animal harboring said population wherein the members of said cell population have an accessible binding site for a ligand, said method comprising the step of

administering to said host a composition comprising an immunogen conjugated to the ligand wherein said immunogen is recognized by an endogenous or an exogenous antibody in the host or is recognized directly by an immune cell in the host; and

administering to said host a therapeutic factor, said factor being selected from the group consisting of a cell killing agent, a tumor penetration enhancer, a chemotherapeutic agent, an antimicrobial agent, a cytotoxic immune cell, and a compound capable of stimulating an endogenous immune response wherein the compound does not bind to the ligand-immunogen conjugate.

48. (Cancelled)

49. (Cancelled)

50. (Previously presented) The pharmaceutical composition of claim 43 wherein the immunogen is a hapten.

51. (Previously presented) The pharmaceutical composition of claim 50 wherein the hapten is fluorescein or dinitrophenyl.

52. (Cancelled)

53. (Previously presented) The method of claim 8 wherein the bonding is covalent bonding through a divalent linker.

54. (Previously presented) The method of claim 8 wherein the bonding is direct covalent bonding.